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Delayed Baroreflex Feedback and Cardiovascular Stability A Testable Science 4.0 Model of Heart-Rate and Blood-Pressure Oscillations

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ABSTRACT

Background: Arterial baroreflex regulation combines haemodynamic dynamics with neural feedback and finite response times. Delayed feedback and pressure oscillations have long been studied, but a reduced model can still be valuable when its assumptions, stability boundary, identifiable quantities, and falsification criteria are made explicit.

Methods: We formulate a two-state linear delay-differential system coupling arterial-pressure deviation to heart-rate deviation. A positive heart-rate-to-pressure pathway and a delayed negative pressure-to-heart-rate pathway generate a scalar loop product. The characteristic equation is analysed for imaginary-axis crossings, and a strictly illustrative parameter set is used to draw the stability boundary and numerical trajectories. The Science 4.0 evidence categories distinguish established physiological context [D], modelling hypotheses [H], conditional deductions [C], and experimental requirements [E].

Results: Under positive relaxation rates a, d and positive loop product $B = ck$, the equilibrium is asymptotically stable for all delays if $B \leq ad$. For $B > ad$, a unique positive crossing frequency and an explicit first critical delay exist. The reduced model loses local stability as the delay passes this boundary. For the illustrative values a = 0.20 s−1, d = 0.30 s−1, and B = 0.24 s−2, the calculated critical delay is 2.536 s and the crossing frequency is 0.0668 Hz. These numbers do not estimate a physiological threshold.

Conclusion: The contribution is a transparent, falsifiable modelling and measurement workflow rather than a claim of a newly discovered cardiovascular mechanism. The framework needs physiological calibration, comparison with competing explanations, and independent validation before biological or clinical interpretation.

Keywords: Baroreflex; Delayed Feedback; Cardiovascular Stability; Blood Pressure; Heart Rate; Delay Differential Equations; System Identification; Science 4.0

Introduction

Arterial-pressure regulation involves baroreceptor sensing, central processing, autonomic efferent pathways, cardiac responses, and changes in vascular tone. Established cardiovascular models account for non-linearity and delays in baroreflex control [1,2]. Oscillatory blood-pressure dynamics, including classical Mayer-wave phenomena, have also been explored using mathematical stability and bifurcation analysis [3]. Consequently, neither the use of delay differential equations nor a delay-induced oscillatory transition is claimed as a novel discovery here. A practical challenge remains: a model can fit spontaneous pressure and heart-rate oscillations without uniquely identifying the physiological mechanism that caused them. The inferential gap is particularly important when respiration, vascular resistance, posture, activity, and other autonomic loops may act as shared inputs. Assessments of spontaneous baroreflex sensitivity require careful interpretation [4,5]. More recent guidance also cautions against treating heart-rate variability (HRV) as a direct measure of sympathetic outflow or sympathovagal balance [6]. This paper proposes a deliberately minimal closed-loop representation intended to make the mechanistic assumptions visible and the mathematical consequences auditable. It is not a full baroreflex model, a patient-specific digital twin, or an experimentally identified transfer function. Its intended value is to specify what could be measured, what might be identifiable, and what would refute the proposed reduced-order explanation.

We use four epistemic labels throughout: [D] published empirical/physiological context; [H] proposed model structure or approximation; [C] derived mathematical or simulated consequence conditional on [H]; [E] independently testable measurement or validation requirement.

Model Definition and Units [H]

Let $p(t) = P(t) - P_0$ denote the departure of an arterial-pressure summary from a chosen steady operating point, measured in mmHg, and let $h(t) = H(t) - H_0$ denote heart-rate departure, measured in beats per minute (bpm). The operating point is assumed approximately constant over the analysis window. Define an external pressure-rate disturbance $\boldsymbol{\upsilon}(t)$ in mmHg s^{-1}. The proposed model is

$$\dot{p}(t) = -ap(t) + ch(t) + u(t),$$
$$\dot{h}(t) = -dh(t) - kP(t-T), \quad (1)$$

where $a > 0$ and $d > 0$ are effective relaxation rates (s^{-1}), $c > 0$ quantifies the local positive influence of a heart-rate deviation on the pressure state (mmHg bpm^{-1} s^{-1}), $k > 0$ is an effective negative pressure-to-heart-rate feedback coefficient (bpm $mmHg^{-1}$ s^{-1}), and $\tau \geq 0$ is a lumped response delay in seconds. Thus $B = ck$ has units s^{-2}. The coefficient k is not directly equivalent to an experimentally reported baroreflex-sensitivity index, because the equation contains an explicit dynamical relaxation term and a delay. The positive c pathway and negative k pathway encode a feedback sign convention, not a complete description of autonomic physiology. Stroke volume, vascular resistance, contractility, respiration, and distributed neural delays are absent. In particular, any inference that heart-rate variation causes all observed pressure changes would be unjustified without independent perturbation or appropriate joint identification. With $\boldsymbol{\upsilon} = 0$, the origin $p = h = 0$ is the equilibrium. For a constant disturbance $\boldsymbol{\upsilon}0$, the shifted equilibrium is $p* = \boldsymbol{\upsilon}_0/(a + B/d)$ and $h_* = -kp_*/d$. Local stability of the shifted linear dynamics is unchanged when the coefficients remain constant (Figure 1).

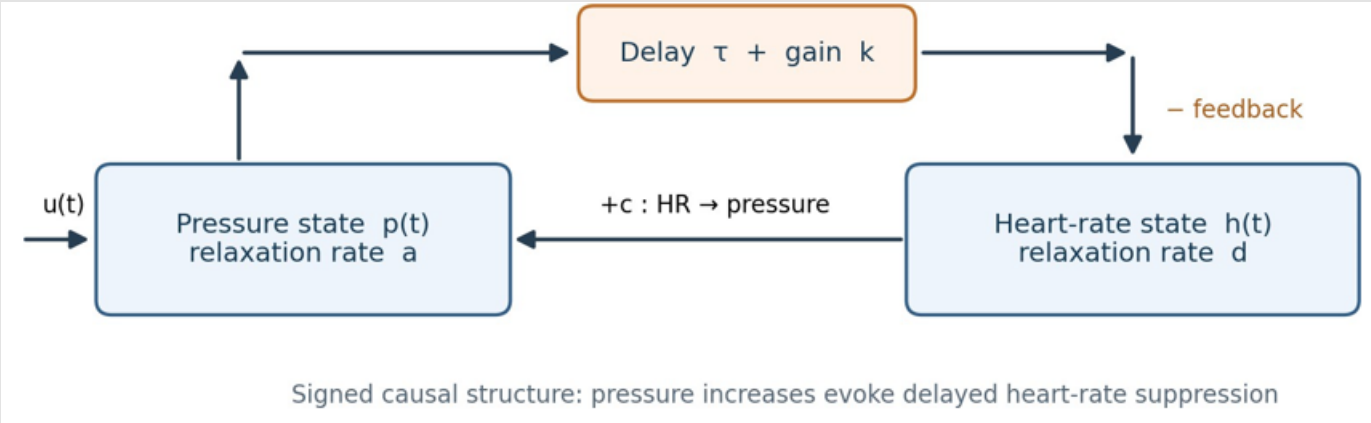


Figure 1: Original reduced-order feedback diagram. The solid pathways express the assumed signs of the two effective couplings. It is a causal model schematic, not anatomical evidence or a clinical circuit diagram.

Stability Analysis [C]

Characteristic Equation

For solutions proportional to $e^{\lambda t}$ and $\boldsymbol{\upsilon} = 0$, the characteristic equation of (1) is

$$F(\lambda, T) = (\lambda + a)(\lambda + d) + Be^{-\lambda T} = 0, \; B = ck > 0. \quad (2)$$

At $\tau = 0$, (2) is $\lambda^2 + (a+d)\lambda + (ad + B) = 0$. Since all coefficients are positive, both characteristic roots have strictly negative real parts. The zero-delay equilibrium is therefore asymptotically stable.

Imaginary-Axis Crossing Condition

Let $\lambda = im$, $m > 0$. Separating the real and imaginary parts of (2) gives $B\cos(mT) = m^2 - ad$, $B\sin(mT) = m(a+d)$. (3)

Squaring and adding yields

$$B^2 = (a^2 + m^2)(d^2 + m^2) \quad (4)$$

The right-hand side increases strictly with $m > 0$ and has its minimum a^2d^2 at zero. Thus a non-zero imaginary crossing is possible if and only if $B > ad$. When $B > ad$, the unique positive crossing frequency is $m_c^2 = \frac{-(a^2+d^2) + \sqrt{(a^2-d^2)^2 + 4B^2}}{2}$ (5)

Taking the phase with a quadrant-aware argument gives the first positive crossing delay $T_c = \frac{a \tan 2(w_c(a+d), w_c^2 - ad)}{w_c}$ (6)

Subsequent imaginary-axis crossings of the same frequency occur at $\tau_n = \tau_c + 2\pi n/m_c$, $n = 1, 2, \ldots$.

Stability Regimes and Direction of Crossing

The mathematical conclusions are:

- **Regime A:** $B \le ad$. No positive-frequency imaginary root exists, and F (0, τ) = $ad+B > 0$. Since the zero-delay problem is stable, the equilibrium remains stable for all finite $\tau \ge 0$ within this linear model.
- **Regime B:** $B > ad$ and $0 \le \tau < \tau_c$. The equilibrium is asymptotically stable.
- **Regime C:** $B > ad$ and $\tau > \tau_c$. At least one characteristic-root pair lies in the right half-plane. The linear equilibrium is unstable.

The imaginary-root crossing at τ_c is transversal and moves toward instability as τ increases. An analytic sign check is given in **Appendix A**. A *linear* instability must not be interpreted as an indefinitely increasing physiological pressure oscillation: nonlinear saturation and other regulatory loops are absent from (1). These thresholds are mathematical properties of the assumed two-state structure. They are not new universal conditions for hypertension, cardiovascular resilience, or a particular patient's prognosis.

Dimensionless Reporting and Frequency-Response Interpretation [C]

For comparison across candidate parameter fits, define the dimensionless loop ratio $G = B/(ad)$ and delay $\Theta = \tau\sqrt{ad}$. The condition $G > 1$ is necessary for a finite-delay stability loss. If $x = m/\sqrt{ad}$ and $\eta = (a + d)/\sqrt{ad}$, equation (4) becomes

$$x^4 + \left(\frac{a}{d}+\frac{d}{a}\right) x^2 + 1 - G^2 = 0 \quad (7)$$

These normalized quantities provide a transparent reporting convention; they are not established physiological biomarkers. The critical delay also depends on the asymmetry between the two relaxation rates, not just on G.

The theoretical disturbance-to-pressure transfer function is

$$G_{pu}(s) = \frac{P(s)}{U(s)} = \frac{s+d}{(s+a)(s+d)+Be^{-sT}} \quad (8)$$

and, assuming zero initial conditions, $H(s) = -ke^{-sT}$ P(s)/(s+d). Equation (8) is a closed- loop transfer function: a spectral peak can reflect feedback, external forcing, or both. Consequently, a peak in the pressure spectrum alone cannot identify k, τ, or a baroreflex origin.

Reproducible Numerical Illustration [C]

We use one explicitly non-empirical parameter configuration solely to check equations (5) and (6) and illustrate the shape of the stability transition: Equation (5) gives m_c = 0.419879 rad s^{-1}. Thus $f_c = m_c/(2\pi)$ = 0.066826 Hz, the corresponding period is 14.964 s, and equation (6) yields τ_c = 2.536244 s. The absolute complex residual of (2) at imc and τ_c is approximately 1.4×10^{-17} in the accompanying numerical check. The crossing frequency is not asserted to match a subject's measured Mayer-wave frequency. To visualize the dynamics, the system was integrated using a fixed-step midpoint method with linear interpolation of the delayed pressure history; the time step was 0.005 s and the initial history was $p(t)$ = 1 mmHg and $h(t)$ = 0 bpm for $t \le 0$. No measurement noise, respiration, or nonlinear saturation was included. Two delays are compared with the same other parameters: τ = 1.0 s (stable) and τ = 3.5 s (unstable). A direct numerical characteristic-root check gives a representative pair $\lambda \approx -0.1203 \pm 0.5017i$ s^{-1} for τ = 1.0 s and $\lambda \approx +0.0308 \pm 0.3687i$ s^{-1} for τ = 3.5 s. The sign of the real part is consistent with the time-domain comparison. These checks establish only numerical reproducibility of the assumed model (Figures 2&3) (Table 1).

Table 1.

Quantity	Illustrative setting	Units
Pressure relaxation a	0.20	s^{-1}
Heart-rate relaxation d	0.30	s^{-1}
HR-to-pressure coupling c	0.40	mmHg / (bpm · s)
Delayed pressure-to-HR coupling k	0.60	bpm/ (mmHg · s)
Product $B = ck$	0.24	s^{-2}
Product ad	0.06	s^{-2}

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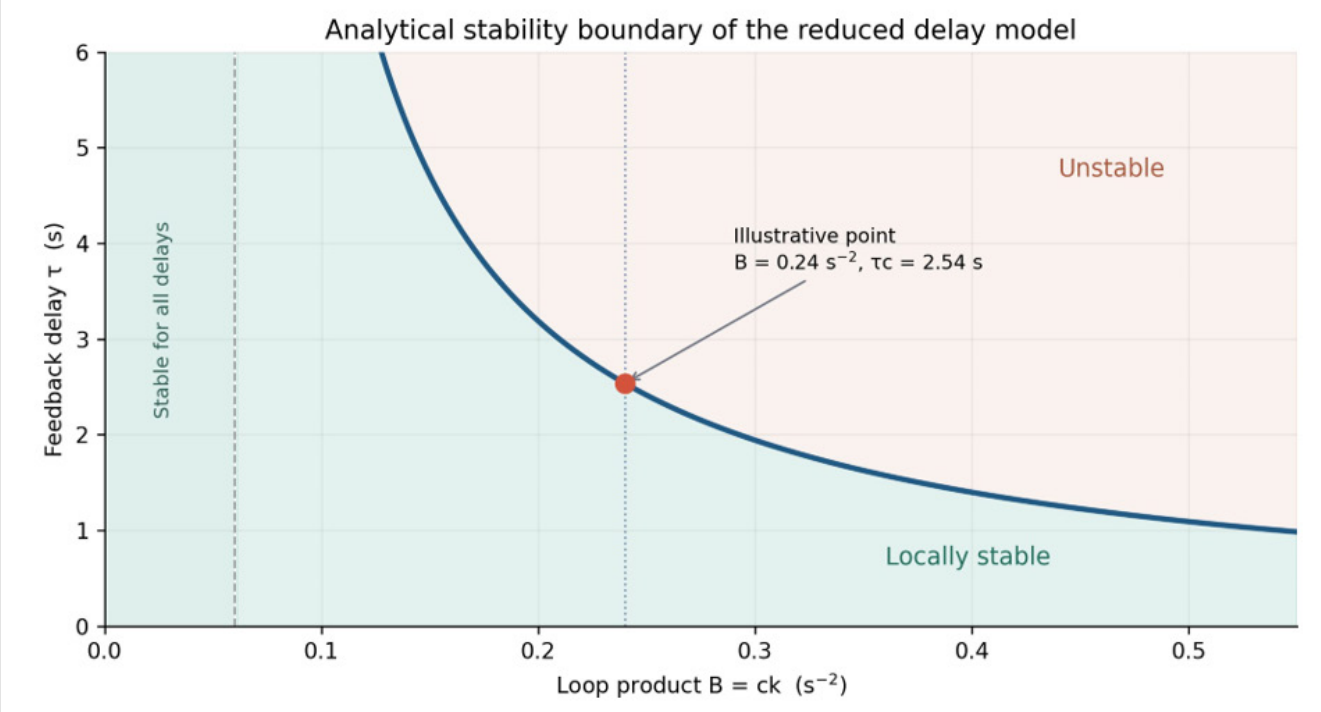


Figure 2: Calculated first stability boundary for fixed $a = 0.20$ and $d = 0.30\ s^{-1}$ while B varies. The shaded regions represent stability properties of equation (1), not clinical risk zones. The marked point is the synthetic parameter set.

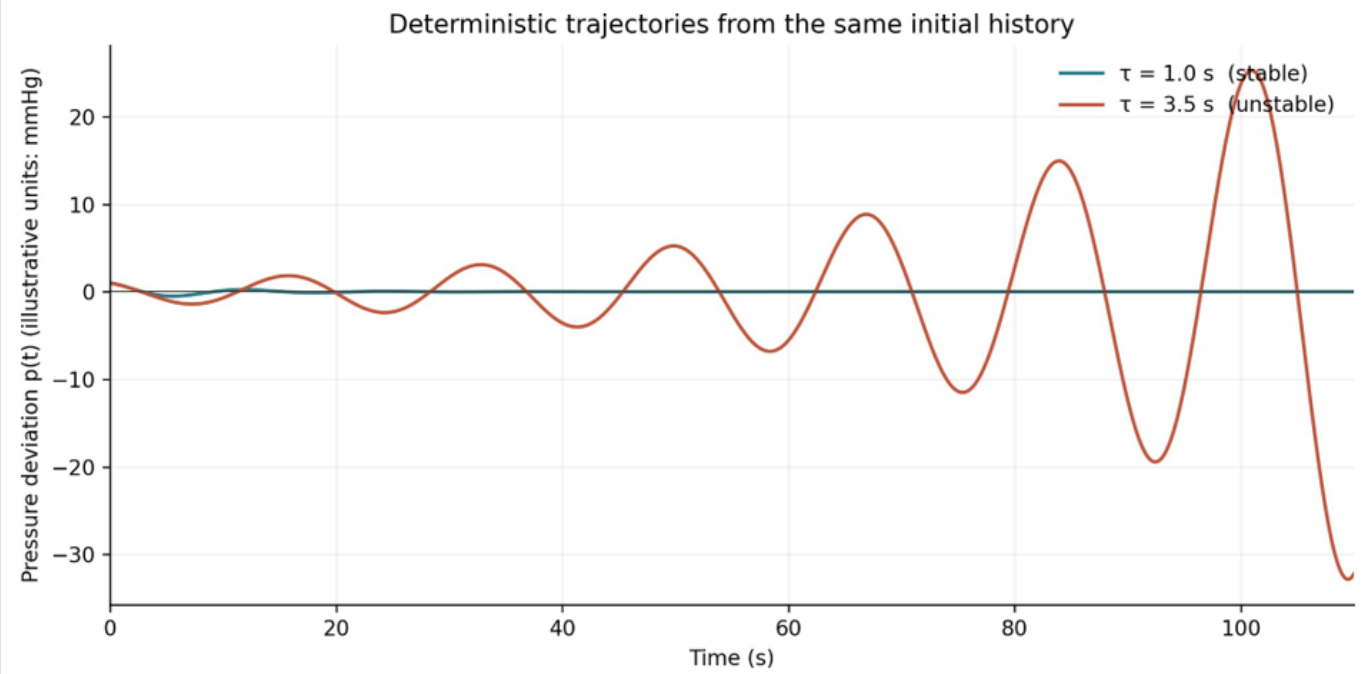


Figure 3: Original deterministic model trajectories with identical initial conditions. The unstable trace grows because equation (1) is linear and lacks saturation; its long-time amplitude is not physiologically meaningful. Both traces are synthetic.

Identifiability and Uncertainty [H/E]

The reduced system is deliberately simple, but its parameters should not be treated as directly observable. If only a pressure time series and its disturbance response are available, (8) depends on c and k principally through their product $B = ck$, so these separate couplings cannot be uniquely recovered from that transfer function alone. With synchronized pressure and heart-rate observations, sufficiently informative perturbations, an explicit observation model, and calibrated measurement scales, identification of additional parameters may become possible. Such practical identifiability must be evaluated rather than presumed. An appropriate estimation model includes observation errors, a possible shared disturbance $\boldsymbol{v}(t)$, uncertain sampling delays, and respiratory covariates. Parameters should be reported with con-fidence or credible intervals and their correlations. The delay estimate must be checked against changes in the observation bandwidth, sampling frequency, smoothing, time alignment, and the use of distributed-delay alternatives. For a parameter vector $\theta = (a, d, c, k, \tau)$, a local sensitivity matrix $J_{ij} = \partial y_i / \partial \theta_j$ may help characterize the inverse problem, but numerical rank under an idealized input does not guarantee identifiability in real recordings. Profile likelihoods, out-of-sample predictions, bootstrap analysis, and explicit comparison with simpler models are important. If reliable pressure and heart-rate perturbation data are unavailable, the model should remain theoretical.

The analysis must also distinguish repeatability, statistical parameter uncertainty, systematic measurement error, model discrepancy, and physiological variability. A narrow confidence interval conditional on an incorrect two-state structure is not evidence that the physiological mechanism has been identified.

Nonequilibrium Perspective: What the Model Does Not Prove [H/C]

A tempting interpretation would be to call HRV a direct entropy dissipator or a cardiovascular energy-saving mechanism. This paper expressly does not make that claim. A rate equation for pressure and heart rate does not itself provide tissue-level energy fluxes, entropy production, or metabolic power. Those quantities would require separate constitutive laws and measurements. HRV is a descriptive statistic of cardiac timing, not a thermodynamic state function [6].

For $\boldsymbol{v} = 0$ and zero delay, one can define the positive quadratic *mathematical storage function*

$$V(p,h) = \frac{1}{2}(kp^2 + ch^2),\ \dot{V} = -akp^2 - cdh^2 \leq 0 \quad (9)$$

For finite delay the derivative becomes

$$\dot{V} = -akp^2 - cdh^2 + ckh(t)[p(t) - p(t-T)] \quad (10)$$

Equations (9&10) provide a control-theoretic illustration of why delay can disrupt a simple dissipation inequality. Importantly, the units of V are not joules under the present variable definitions. It must not be interpreted as physiological free energy, ATP consumption, entropy, or an experimentally calibrated dissipation potential. A genuine non-equilibrium thermodynamic extension would need additional measured variables (e.g., flow, resistance, compliance, cardiac work, and metabolic expenditure) and a dimensionally consistent energy balance.

Falsifiable Predictions and Experimental Programme [E]

Prediction P1 - Delay Sensitivity: If the two-state negative-feedback approximation de-scribes the dominant pressure–heart-rate coupling over a specified operating window, then changes in effective feedback delay and loop gain should alter spectral phase, impulse response, and damp-ing in directions compatible with equations (2,6). *Potential falsification:* estimated responses or intervention effects are incompatible with the fitted signs or delay dependence after controlling for shared inputs.

Prediction P2 - Cross-Channel Identifiability: Joint pressure and heart-rate measurements under sufficiently informative input variation should constrain the direction and magnitude of the effective couplings better than pressure-only data. *Potential falsification:* stable, reproducible separate parameter estimates cannot be obtained or remain non-identifiable even with the planned stimuli.

Prediction P3 - Competing-Model Comparison: A fitted delayed-feedback model should outperform justified simpler benchmarks on held-out pressure and heart-rate trajectories if its delayed pathway is genuinely needed. Benchmarks include a no-delay model, an externally forced oscillator, a common-input autoregressive model, and a distributed-delay or vascular-resistance model. *Potential falsification:* the delayed model offers no predictive advantage, or a competing mechanism explains the data with fewer assumptions.

A prospective experimental programme should specify:

- **(i)** Ethical approval and informed consent for human recordings where applicable;
- **(ii)** Continuous beat-to-beat arterial-pressure measurement and a synchronized ECG or validated heart-rate signal;
- **(iii)** Respiratory phase and breathing rate;
- **(iv)** Activity, posture, medication and relevant physiological covariates;
- **(v)** Calibrated time synchronization; and
- **(vi)** Independent validation windows.

Non-invasive or clinically standard perturbation protocols would need to be chosen and administered by qualified investigators, not inferred from this paper. Before collecting data, investigators should pre-register measurable endpoints and analysis bands, define param-

eter priors or bounds, set model-selection criteria, describe handling of ectopic beats and artefacts, and specify uncertainty propagation. Respiratory confounding must be treated ex-plicitly; coherence alone is insufficient to establish causal feedback [4,5]. No proposed manoeuvre should be adopted on the basis of this theoretical model without clinical and ethical review.

Discussion

This mathematical exercise illustrates a basic but often overlooked distinction between a mechanistically interpretable equation and a demonstrated physiological mechanism. The closed loop of equation (1) produces clear stability predictions: at fixed relaxation rates, sufficiently strong delayed negative feedback can be locally destabilizing. This finding aligns in broad conceptual terms with prior delayed-feedback and gain-induced oscillation models [1-3], and therefore should not be portrayed as unprecedented. The specific contribution of the present manuscript is a compact, auditable set of equations tied to a measurement and falsification plan and to an explicit epistemic separation of assumptions, calculations, and experiments. Before any originality claim is made, a complete literature com-parison and independent specialist review should assess whether this presentation provides a substantive methodological advance beyond established baroreflex models. Several limitations are decisive. First, changes in arterial pressure can be driven by peripheral resistance and stroke volume rather than heart rate alone. Second, a single pure delay is a poor substitute for heterogeneous neural conduction and effector time constants. Third, observed spontaneous pressure–heart-rate coupling may be affected by respiration and external common drivers. Fourth, nonlinearity, saturation, and operating-point dependence become essential near or beyond instability.

Fifth, instantaneous HRV metrics are not valid surrogate measurements of loop parameters or thermodynamic entropy production [6]. Finally, the model's illustrative coefficients and derived threshold have not been estimated from humans, animals, or devices. The next scientific step is not to choose a more attractive simulation but to decide which measurements would discriminate the proposed feedback mechanism from plausible alternatives. Only then would a biological or translational interpretation be warranted.

Conclusion

A minimal delayed baroreflex representation yields a dimensionally explicit characteristic equation and a testable mathematical stability boundary. Under its assumptions, $B \leq ad$ ensures delay-independent linear stability, while $B > ad$ permits a first stability loss at the delay τ_c given by equation (6). The figures are reproducible theoretical illustrations, not empirical findings. The framework offers a candidate structure for future systems-identification work but does not establish new physiological causation, measured thermodynamic dissipation, disease prediction, or clinical efficacy.

Declarations

Study Design: Theoretical analysis and deterministic numerical illustration; no experimental study performed.

Data Availability: No participant or experimental dataset was generated or analyzed. The figure-generation script and illustrative parameter settings accompany this manuscript.

Ethics: No human participants or animals were involved in this theoretical work. Proposed future studies would require their own approvals.

Conflicts of Interest: The author declares no conflicts of interest.

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Authorship and Attribution: Julien Boblique is the sole author listed on this manuscript. The author is responsible for checking its content, verifying appropriate attribution, and approving the final submitted version.

Status of Originality: The equations use classical linear delay-systems reasoning and are not presented as a new general theorem. Journal suitability, novelty, and bibliographic completeness require independent checking.

References

1. Ottesen JT (1997) Modelling of the baroreflex-feedback mechanism with time-delay. Journal of Mathematical Biology 36(1): 41-63.
2. Ottesen JT (2000) Modelling the dynamical baroreflex-feedback control. Mathematical and Com-puter Modelling 31(4-5): 167-173.
3. Abbiw Jackson RM, Langford WF (1998) Gain-induced oscillations in blood pressure. Journal of Mathematical Biology 37(3): 203-234.
4. Parati G, Di Rienzo M, Mancia G (2000) How to measure baroreflex sensitivity: from the cardiovas-cular laboratory to daily life. Journal of Hypertension 18(1): 7-19.
5. Di Rienzo M, Castiglioni P, Mancia G, Parati G, Pedotti A (1997) Critical appraisal of indices for the assessment of baroreflex sensitivity. Methods of Information in Medicine 36(4-5): 246-249.
6. Carter JR, Jenkins NDM, Bigalke JA, Austin T Robinson, Manda L Keller Ross, et al. (2026) Guidelines for rigor and reproducib-ility of heart rate variability within human cardiovascular research. American Journal of Physiology-Heart and Circulatory Physiology 331(3): H918-H943.

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